27nov01 17:42:00 User217743 Session D538.1
$0.00 0.203 DialUnits FileHomeBase
$0.00 Estimated cost FileHomeBase
$0.00 Estimated cost this search
$0.00 Estimated total session cost 0.203 DialUnits
File 410:Chronolog(R) 1981-2001/Nov
(c) 2001 The Dialog Corporation

Set Items Description
--- ----- ----------
? set hi *;set hi *

HILIGHT set on as '*'*
HILIGHT set on as ''
? b 411

27nov01 17:42:05 User217743 Session D538.2
$0.00 0.065 DialUnits File410
$0.00 Estimated cost File410
$0.00 Estimated cost this search
$0.00 Estimated total session cost 0.268 DialUnits
File 411:DIALINDEX(R)

DIALINDEX(R)
(c) 2001 The Dialog Corporation plc

*** DIALINDEX search results display in an abbreviated
*** *** format unless you enter the SET DETAIL ON
command. *** ? set files biochem

>>> 162 is unauthorized
>>>1 of the specified files is not available
You have 25 files in your file list.
(To see banners, use SHOW FILES command)
? s ((il or interleukin)()22 or (ztgf or
transforming()growth()factor)()beta()9)

Your SELECT statement is:
s ((il or interleukin)()22 or (ztgf or
transforming()growth()factor)()beta()9)

Items File
----- ----
8 5: Biosis Previews(R)_1969-2001/Nov W3
1 6: NTIS_1964-2001/Dec W2
17 34: SciSearch(R) Cited Ref
Sci_1990-2001/Nov W4 1 50: CAB
Abstracts_1972-2001/Oct
6 71: ELSEVIER BIOBASE_1994-2001/Nov
W4
8 73: EMBASE_1974-2001/Nov W3
6 76: Life Sciences Collection_1982-2001/Nov
1 94: JICST-EPlus_1985-2001/Oct W3
1 98: General Sci
Abs/Full-Text_1984-2001/Oct 2 103: Energy
SciTec_1974-2001/Sep B2
4 143: Biol. & Agric. Index_1983-2001/Sep
6 144: Pascal_1973-2001/Nov W4
16 155: MEDLINE(R)_1966-2001/Dec W4
7 156: ToxFile_1966-2001/Oct W3
1 172: EMBASE Alert_2001/Nov W4
8 399: CA SEARCH(R)_1967-2001/UD=13522

16 files have one or more items; file list includes 25
files.
? rf

Your last SELECT statement was:
S ((IL OR INTERLEUKIN)()22 OR (ZTGF OR
TRANSFORMING()GROWTH()FACTOR)()B- ETA()9)
Ref Items File
--- ----- ----
N1 17 34: SciSearch(R) Cited Ref
Sci_1990-2001/Nov W4 N2 16 155:
MEDLINE(R)_1966-2001/Dec W4
N3 8 5: Biosis Previews(R)_1969-2001/Nov W3
N4 8 73: EMBASE_1974-2001/Nov W3
N5 8 399: CA SEARCH(R)_1967-2001/UD=13522
N6 7 156: ToxFile_1966-2001/Oct W3
N7 6 71: ELSEVIER BIOBASE_1994-2001/Nov
W4
N8 6 76: Life Sciences
Collection_1982-2001/Nov N9 6 144:
Pascal_1973-2001/Nov W4
N10 4 143: Biol. & Agric. Index_1983-2001/Sep
16 files have one or more items; file list includes 25 files.

- Enter P or PAGE for more -
? b n2,n1,94

27nov01 17:46:10 User217743 Session D538.3
$3.40 2.719 DialUnits File411
$3.40 Estimated cost File411
$0.25 TYMNET
$3.65 Estimated cost this search
$3.65 Estimated total session cost 2.988 DialUnits
SYSTEM:OS - DIALOG OneSearch
File 155:MEDLINE(R) 1966-2001/Dec W4
File 34:SciSearch(R) Cited Ref Sci 1990-2001/Nov W4
(c) 2001 Inst for Sci Info
File 94:JICST-EPlus 1985-2001/Oct W3
(c)2001 Japan Science and Tech Corp(JST)
*File 94: There is no data missing. UDs have been
adjusted to reflect the current months data. See Help
News94 for details.

Set Items Description
--- ----- ----------
? s ((il or interleukin)()22 or (ztgf or
transforming()growth()factor)()beta()9)
189230 IL
231023 INTERLEUKIN

450715 22
27 (IL OR INTERLEUKIN)(W)22
0 ZTGF
76398 TRANSFORMING
1687589 GROWTH
1310883 FACTOR
52355
TRANSFORMING(W)GROWTH(W)FACTOR
962242 BETA
1279765 9
7 (ZTGF OR
TRANSFORMING(W)GROWTH(W)FACTOR)(W)BETA(W)
9 S1 34 ((IL OR INTERLEUKIN)()22 OR (ZTGF
OR

TRANSFORMING()GROWTH()FACTOR)()BETA()9)
? rd

...completed examining records
S2 27 RD (unique items)
? t s2/3,ab,kwic/all

2/3,AB,KWIC/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11690678 21448676 PMID: 11564763
Cutting edge: STAT activation by IL-19, IL-20 and mda-7 through IL-20 receptor complexes of two types.
Dumoutier L; Leemans C; Lejeune D; Kotenko SV; Renauld JC Ludwig Institute for Cancer Research, Brussels Branch, Avenue Hippocrate 74, B-1200 Brussels, Belgium.
Journal of immunology (United States) Oct 1 2001, 167 (7) p3545-9, ISSN 0022-1767 Journal Code: IFB
Contract/Grant No.: RO1 AI51139, AI, NIAID
Languages: ENGLISH
Document type: Journal Article
Record type: In Process
IL-10-related cytokines include IL-20 and *IL*-*22*, which induce, respectively, keratinocyte proliferation and acute-phase production by hepatocytes, as well as IL-19, melanoma differentiation-associated gene 7, and AK155, three cytokines for which no activity nor receptor complex has been described thus far. Here, we show that mda-7 and IL-19 bind to the previously described IL-20R complex, composed by cytokine receptor family 2-8/IL-20Ralpha and DIRS1/IL-20Rbeta (type I IL-20R). In addition, mda-7 and IL-20, but not IL-19, bind to another receptor complex, composed by IL-22R and DIRS1/IL20Rbeta (type II IL-20R). In both cases, binding of the ligands results in STAT3 phosphorylation and activation of a minimal promoter including STAT-binding sites. Taken together, these results demonstrate that: 1) IL-20 induces STAT activation through IL-20R complexes of two types; 2) mda-7 and IL-20 redundantly signal through both complexes; and 3) IL-19 signals only through the type I IL-20R complex.
IL-10-related cytokines include IL-20 and *IL*-*22*, which induce, respectively, keratinocyte proliferation and acute phase production by hepatocytes, as well as IL-19, melanoma differentiation-associated gene 7, and AK155, three cytokines...

2/3,AB,KWIC/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11662350 21396522 PMID: 11481447
A soluble class II cytokine receptor, IL-22RA2, is a naturally occurring *IL*-*22* antagonist.
Xu W; Presnell SR; Parrish-Novak J; Kindsvogel W; Jaspers S; Chen Z; Dillon SR; Gao Z; Gilbert T; Madden K; Schlutsmeyer S; Yao L; Whitmore TE; Chandrasekher Y; Grant FJ; Maurer M; Jelinek L; Storey H; Brender T; Hammond A; Topouzis S; Clegg CH; Foster DC
ZymoGenetics Inc., Seattle, WA 98102, USA.
Proceedings of the National Academy of Sciences of the United States of America (United States) Aug 14 2001, 98 (17) p9511-6, ISSN 0027-8424 Journal Code: PV3
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
IL-*22* is an IL-10 homologue that binds to and signals through the class II cytokine receptor heterodimer IL-22RA1/CRF2-4. *IL*- *22* is produced by T cells and induces the production of acute-phase reactants in vitro and in vivo, suggesting its involvement in inflammation. Here we report the identification of a class II cytokine receptor designated IL-22RA2 (*IL*-*22* receptor-alpha 2) that appears to be a naturally expressed soluble receptor. IL-22RA2 shares amino acid sequence homology with IL-22RA1 (also known as IL-22R, zcytor11, and CRF2-9) and is physically adjacent to IL-20Ralpha and IFN-gammaR1 on chromosome 6q23.3-24.2. We demonstrate that IL-22RA2 binds specifically to *IL*- *22* and neutralizes *IL*-*22*-induced proliferation of BaF3 cells expressing *IL*-*22* receptor subunits. IL-22RA2 mRNA is highly expressed in placenta and spleen by Northern blotting. PCR analysis using RNA from various tissues and cell lines showed that IL-22RA2 was expressed in a range of tissues, including those in the digestive, female reproductive, and immune systems. In situ hybridization revealed the dominant cell types expressing IL-22RA2 were mononuclear cells and epithelium. Because *IL*-*22* induces the expression of acute phase reactants, IL-22RA2 may play an important role as an *IL*-*22* antagonist in the regulation of inflammatory responses.

A soluble class II cytokine receptor, IL-22RA2, is a

naturally occurring *IL*-*22* antagonist.
IL-*22* is an IL-10 homologue that binds to and signals through the class II cytokine receptor heterodimer IL-22RA1/CRF2-4. *IL*- *22* is produced by T cells and induces the production of acute-phase reactants in vitro and in vivo, suggesting its involvement in inflammation. Here we report the identification of a class II cytokine receptor designated IL-22RA2 (*IL*-*22* receptor-alpha 2) that appears to be a naturally expressed soluble receptor. IL-22RA2 shares amino acid sequence homology with IL-22RA1 (also known as...

... 9) and is physically adjacent to IL-20Ralpha and IFN-gammaR1 on chromosome 6q23.3-24.2. We demonstrate that IL-22RA2 binds specifically to *IL*-*22* and neutralizes *IL*-*22*-induced proliferation of BaF3 cells expressing *IL*-*22* receptor subunits. IL-22RA2 mRNA is highly expressed in placenta and spleen by Northern blotting. PCR analysis using RNA from various tissues and cell lines...

... in the digestive, female reproductive, and immune systems. In situ hybridization revealed the dominant cell types expressing IL-22RA2 were mononuclear cells and epithelium. Because *IL*-*22* induces the expression of acute phase reactants, IL-22RA2 may play an important role as an *IL*-*22* antagonist in the regulation of inflammatory responses.
Chemical Name: Interleukins; Neoplasm Proteins; RNA, Messenger; Receptors, Interleukin; Recombinant Fusion Proteins; *interleukin*- *22*; *interleukin*-*22* receptor

2/3,AB,KWIC/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11574279 21286453 PMID: 11390454
Identification, cloning, and characterization of a novel soluble receptor that binds *IL*-*22* and neutralizes its activity. Kotenko SV; Izotova LS; Mirochnitchenko OV; Esterova E; Dickensheets H; Donnelly RP; Pestka S
Department of Molecular Genetics and Microbiology, University of Medicine and Dentistry, Robert Wood Johnson Medical School, Piscataway, NJ 08854, USA. kotenkse@umdnj.edu
Journal of immunology (United States) Jun 15 2001, 166 (12) p7096-103, ISSN 0022-1767 Journal Code: IFB
Contract/Grant No.: 1P30-CA72720, CA, NCI; RO1 AI36450, AI, NIAID; RO1 AI43369, AI, NIAID; RO1-CA46465, CA, NCI
Languages: ENGLISH
Document type: Journal Article
Record type: Completed

With the use of a partial sequence of the human genome, we identified a gene encoding a novel soluble receptor belonging to the class II cytokine receptor family. This gene is positioned on chromosome 6 in the vicinity of the IFNGR1 gene in a head-to-tail orientation. The gene consists of six exons and encodes a 231-aa protein with a 21-aa leader sequence. The secreted mature protein demonstrates 34% amino acid identity to the extracellular domain of the IL-22R1 chain. Cross-linking experiments demonstrate that the protein binds *IL*-*22* and prevents binding of *IL*-*22* to the functional cell surface IL-22R complex, which consists of two subunits, the IL-22R1 and the IL-10R2c chains. Moreover, this soluble receptor, designated *IL*-*22*-binding protein (BP), is capable of neutralizing *IL*-*22* activity. In the presence of the IL-22BP, *IL*-*22* is unable to induce Stat activation in *IL*-*22* -responsive human lung carcinoma A549 cells. IL-22BP also blocked induction of the suppressors of cytokine signaling-3 (SOCS-3) gene expression by *IL*-*22* in HepG2 cells. To further evaluate IL-22BP action, we used hamster cells expressing a modified IL-22R complex consisting of the intact IL-10R2c and the chimeric IL-22R1/gammaR1 receptor in which the IL-22R1 intracellular domain was replaced with the IFN-gammaR1 intracellular domain. In these cells, *IL*-*22* activates biological activities specific for IFN-gamma, such as up-regulation of MHC class I Ag expression. The addition of IL-22BP neutralizes the ability of *IL*-*22* to induce Stat activation and MHC class I Ag expression in these cells. Thus, the soluble receptor designated IL-22BP inhibits *IL*-*22* activity by binding *IL*-*22* and blocking its interaction with the cell surface IL-22R complex.

Identification, cloning, and characterization of a novel soluble receptor that binds *IL*-*22* and neutralizes its activity. ... secreted mature protein demonstrates 34% amino acid identity to the extracellular domain of the IL-22R1 chain. Cross-linking experiments demonstrate that the protein binds *IL*-*22* and prevents binding of *IL*-*22* to the functional cell surface IL-22R complex, which consists of two subunits, the IL-22R1 and the IL-10R2c chains. Moreover, this soluble receptor, designated *IL*-*22*-binding protein (BP), is capable of neutralizing *IL*-*22* activity. In the presence of the IL-22BP, *IL*-*22* is unable to induce Stat activation in *IL*-*22* -responsive human lung carcinoma A549 cells. IL-22BP also blocked induction of the suppressors of cytokine signaling-3 (SOCS-3) gene expression by *IL*-*22* in HepG2 cells. To further evaluate IL-22BP action, we used hamster cells expressing a modified IL-22R complex consisting of the intact IL-10R2c and the chimeric IL-22R1/gammaR1 receptor in which the IL-22R1 intracellular domain was replaced with the IFN-gammaR1 intracellular domain. In these cells,

IL-*22* activates biological activities specific for IFN-gamma, such as up-regulation of MHC class I Ag expression. The addition of IL-22BP neutralizes the ability of *IL*-*22* to induce Stat activation and MHC class I Ag expression in these cells. Thus, the soluble receptor designated IL-22BP inhibits *IL*-*22* activity by binding *IL*-*22* and blocking its interaction with the cell surface IL-22R complex.

Chemical Name: Carrier Proteins; DNA, Complementary; *IL*-*22* binding protein; Interleukins; Ligands; Receptors, Interleukin; *interleukin*-*22*; *interleukin*-*22* receptor

2/3,AB,KWIC/4 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11574278 21286452 PMID: 11390453

Cloning and characterization of *IL*-*22* binding protein, a natural antagonist of IL-10-related T cell-derived inducible factor/ *IL*-*22*.

Dumoutier L; Lejeune D; Colau D; Renauld JC

Ludwig Institute for Cancer Research, Brussels Branch and the Experimental Medicine Unit, Christian de Duve Institute of Cellular Pathology, Universite de Louvain, Brussels, Belgium.

Journal of immunology (United States) Jun 15 2001, 166 (12) p7090-5, ISSN 0022-1767 Journal Code: IFB

Languages: ENGLISH

Document type: Journal Article

Record type: Completed

The class II cytokine receptor family includes the receptors for IFN-alphabeta, IFN-gamma, IL-10, and IL-10-related T cell-derived inducible factor/*IL*-*22*. By screening genomic DNA databases, we identified a gene encoding a protein of 231 aa, showing 33 and 34% amino acid identity with the extracellular domains of the *IL*-*22* receptor and of the IL-20R/cytokine receptor family 2-8, respectively, but lacking the transmembrane and cytoplasmic domains. A lower but significant sequence identity was found with other members of this family such as the IL-10R (29%), cytokine receptor family 2-4/IL-10Rbeta (30%), tissue factor (26%), and the four IFN receptor chains (23-25%). This gene is located on chromosome 6q24, at 35 kb from the IFNGR1 gene, and is expressed in various tissues with maximal expression in breast, lungs, and colon. The recombinant protein was found to bind IL-10-related T cell-derived inducible factor/*IL*-*22* , and to inhibit the activity of this cytokine on hepatocytes and intestinal epithelial cells. We propose to name this natural cytokine antagonist IL-22BP for *IL*-*22* binding protein.

Cloning and characterization of *IL*-*22* binding protein, a natural antagonist of IL-10-related T cell-derived inducible factor/ *IL*-*22*.

The class II cytokine receptor family includes the receptors for IFN-alphabeta, IFN-gamma, IL-10, and IL-10-related T cell-derived inducible factor/*IL*-*22*. By screening genomic DNA databases, we identified a gene encoding a protein of 231 aa, showing 33 and 34% amino acid identity with the extracellular domains of the *IL*-*22* receptor and of the IL-20R/cytokine receptor family 2-8, respectively, but lacking the transmembrane and cytoplasmic domains. A lower but significant sequence identity...

...various tissues with maximal expression in breast, lungs, and colon. The recombinant protein was found to bind IL-10-related T cell-derived inducible factor/*IL*-*22* , and to inhibit the activity of this cytokine on hepatocytes and intestinal epithelial cells. We propose to name this natural cytokine antagonist IL-22BP for *IL*-*22* binding protein.

Chemical Name: Carrier Proteins; Cytokines; IL-10-related T cell-derived inducible factor, IL-TIF; *IL*-*22* binding protein; Interleukins; Receptors, Cytokine; Receptors, Interleukin; *interleukin*-*22*; *interleukin*-*22* receptor; Interleukin-10

2/3,AB,KWIC/5 (Item 5 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11376341 21264727 PMID: 11035029

Identification of the functional *interleukin*-*22* (*IL*- *22*) receptor complex: the IL-10R2 chain (IL-10Rbeta) is a common chain of both the IL-10 and *IL*-*22* (IL-10-related T cell-derived inducible factor, IL-TIF) receptor complexes.

Kotenko SV; Izotova LS; Mirochnitchenko OV; Esterova E; Dickensheets H; Donnelly RP; Pestka S

Department of Molecular Genetics and Microbiology, Robert Wood Johnson Medical School, Piscataway, New Jersey 08854-5635, USA. kotenkse@umdnj.edu Journal of biological chemistry (United States) Jan 26 2001, 276 (4) p2725-32, ISSN 0021-9258 Journal Code: HIV

Contract/Grant No.: 1P30-CA72720, CA, NCI; RO1-AI36450, AI, NIAID; RO1-AI43369, AI, NIAID; RO1-CA46465, CA, NCI

Languages: ENGLISH

Document type: Journal Article

Record type: Completed

Interleukin-10 (IL-10)-related T cell-derived inducible factor (IL-TIF; provisionally designated *IL*-*22*) is a cytokine with limited homology to IL-10. We report here the identification of a functional IL-TIF receptor complex that consists of two receptor chains, the orphan CRF2-9 and IL-10R2, the second chain of the IL-10 receptor complex. Expression of the CRF2-9 chain in monkey COS cells renders them sensitive to IL-TIF. However, in hamster cells both chains, CRF2-9 and

IL-10R2, must be expressed to assemble the functional IL-TIF receptor complex. The CRF2-9 chain (or the IL-TIF-R1 chain) is responsible for Stat recruitment. Substitution of the CRF2-9 intracellular domain with the IFN-gammaR1 intracellular domain changes the pattern of IL-TIF-induced Stat activation. The CRF2-9 gene is expressed in normal liver and kidney, suggesting a possible role for IL-TIF in regulating gene expression in these tissues. Each chain, CRF2-9 and IL-10R2, is capable of binding IL-TIF independently and can be cross-linked to the radiolabeled IL-TIF. However, binding of IL-TIF to the receptor complex is greater than binding to either receptor chain alone. Sharing of the common IL-10R2 chain between the IL-10 and IL-TIF receptor complexes is the first such case for receptor complexes with chains belonging to the class II cytokine receptor family, establishing a novel paradigm for IL-10-related ligands similar to the shared use of the gamma common chain (gamma(c)) by several cytokines, including IL-2, IL-4, IL-7, IL-9, and IL-15.

Identification of the functional *interleukin*-*22* (*IL*- *22*) receptor complex: the IL-10R2 chain (IL-10Rbeta) is a common chain of both the IL-10 and *IL*-*22* (IL-10-related T cell-derived inducible factor, IL-TIF) receptor complexes.

Interleukin-10 (IL-10)-related T cell-derived inducible factor (IL-TIF; provisionally designated *IL*-*22*) is a cytokine with limited homology to IL-10. We report here the identification of a functional IL-TIF receptor complex that consists of two...

Chemical Name: Cross-Linking Reagents; Cytokines; IL-10-related T cell-derived inducible factor, IL-TIF; Interleukins; Ligands; Receptors, Interleukin; interleukin-10 receptor; *interleukin*-*22*; *interleukin*-*22* receptor

2/3,AB,KWIC/6 (Item 6 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11259213 21214440 PMID: 11313978

Smads mediate signaling of the TGFbeta superfamily in normal keratinocytes but are lost during skin chemical carcinogenesis. He W; Cao T; Smith DA; Myers TE; Wang XJ

Department of Dermatology, Baylor College of Medicine, Houston, Texas, TX 77030, USA.

Oncogene (England) Jan 25 2001, 20 (4) p471-83, ISSN 0950-9232 Journal Code: ONC

Contract/Grant No.: CA 87849, CA, NCI; CA79998, CA, NCI

Languages: ENGLISH

Document type: Journal Article

Record type: Completed

The Smads are the signaling mediators of the TGFbeta superfamily. In the present study, we examined Smad expression in mouse epidermis and chemically-induced skin tumors. Mutations in Smad2 and -4 genes were also screened. Transcripts of Smad1 through -5 were constantly expressed in the epidermis regardless of changes in TGFbeta signaling, state of differentiation and stages of carcinogenesis. Smad7 transcripts were barely detectable in keratinocytes, but were induced by TGFbeta1 treatment and in chemically-induced skin tumors. At the protein level, Smad1 was detected throughout the epidermis, whereas Smad2 through -5 exhibited greater levels in suprabasal layers than basal keratinocytes. In cultured keratinocytes, Smad2, -3 and -4 underwent nuclear translocation upon TGFbeta1 treatment. Furthermore, nuclear translocation of Smads correlated with decreased BrdU labeling in proliferative keratinocytes. Although no mutations were detected in the Smad2 and -4 genes in tumors, proteins of Smad1 through -5 were partially or completely lost in carcinomas. These data document that Smads are expressed at high levels in the epidermis and mediate signaling of the TGFbeta superfamily. During skin carcinogenesis, loss of Smad1 through -5 and overexpression of Smad7 may contribute to the loss of growth inhibition mediated by TGFbeta superfamily members, thus resulting in tumor progression.

Chemical Name: Carcinogens; RNA, Messenger; RNA, Neoplasm; Transcription Factors; *Transforming* *Growth* *Factor* *beta*; *9* ,10-Dimethyl-1,2-benzanthracene

2/3,AB,KWIC/7 (Item 7 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11216676 21069354 PMID: 11197690

IL-TIF/*IL*-*22* : genomic organization and mapping of the human and mouse genes.

Dumoutier L; Van Roost E; Ameye G; Michaux L; Renauld JC Ludwig Institute for Cancer Research, Brussels Branch, Experimental Medicine Unit, Christian de Duve Institute of Cellular Pathology, Brussels, Belgium.

Genes and immunity (England) Dec 2000, 1 (8) p488-94, ISSN 1466-4879 Journal Code: DXO

Languages: ENGLISH

Document type: Journal Article

Record type: Completed

IL-TIF is a new cytokine originally identified as a gene induced by IL-9 in murine T lymphocytes, and showing 22% amino acid identity with IL-10. Here, we report the sequence and organization of the mouse and human IL-TIF genes, which both consist of 6 exons spreading over approximately 6 Kb. The IL-TIF gene is a single copy gene in humans, and is located on chromosome 12q15, at 90 Kb from the IFN gamma gene, and at 27 Kb from the

AK155 gene, which codes for another IL-10-related cytokine. In the mouse, the IL-TIF gene is located on chromosome 10, also in the same region as the IFN gamma gene. Although it is a single copy gene in BALB/c and DBA/2 mice, the IL-TIF gene is duplicated in other strains such as C57Bl/6, FVB and 129. The two copies, which show 98% nucleotide identity in the coding region, were named IL-TIF alpha and IL-TIF beta. Beside single nucleotide variations, they differ by a 658 nucleotide deletion in IL-TIF beta, including the first non-coding exon and 603 nucleotides from the promoter. A DNA fragment corresponding to this deletion was sufficient to confer IL-9-regulated expression of a luciferase reporter plasmid, suggesting that the IL-TIF beta gene is either differentially regulated, or not expressed at all.

IL-TIF/*IL*-*22* : genomic organization and mapping of the human and mouse genes.

Chemical Name: Cytokines; IL-10-related T cell-derived inducible factor, IL-TIF; Interleukins; *interleukin*-*22*

2/3,AB,KWIC/8 (Item 8 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10836687 20469498 PMID: 10875937

Interleukin (*IL*)-*22* , a novel human cytokine that signals through the interferon receptor-related proteins CRF2-4 and IL-22R. Xie MH; Aggarwal S; Ho WH; Foster J; Zhang Z; Stinson J; Wood WI; Goddard AD; Gurney AL

Department of Molecular Biology, Genentech, Inc., South San Francisco, California 94080, USA.

Journal of biological chemistry (UNITED STATES) Oct 6 2000, 275 (40) p31335-9, ISSN 0021-9258 Journal Code: HIV

Languages: ENGLISH

Document type: Journal Article

Record type: Completed

We report the identification of a novel human cytokine, distantly related to interleukin (IL)-10, which we term *IL*-*22*. *IL*-*22* is produced by activated T cells. *IL*-*22* is a ligand for CRF2-4, a member of the class II cytokine receptor family. No high affinity ligand has yet been reported for this receptor, although it has been reported to serve as a second component in IL-10 signaling. A new member of the interferon receptor family, which we term IL-22R, functions as a second component together with CRF2-4 to enable *IL*-*22* signaling. *IL*-*22* does not bind the IL-10R. Cell lines were identified that respond to *IL*-*22* by activation of STATs 1, 3, and 5, but were unresponsive to IL-10. In contrast to IL-10, *IL*-*22* does not inhibit the production of proinflammatory cytokines by monocytes in response to LPS nor does it impact IL-10 function on monocytes, but it has modest inhibitory effects on IL-4 production from Th2 T cells.

Interleukin (*IL*)-*22* , a novel human cytokine that signals through the interferon receptor-related proteins CRF2-4 and IL-22R. We report the identification of a novel human cytokine, distantly related to interleukin (IL)-10, which we term *IL*-*22*. *IL*-*22* is produced by activated T cells. *IL*-*22* is a ligand for CRF2-4, a member of the class II cytokine receptor family. No high affinity ligand has yet been reported for this...

...signaling. A new member of the interferon receptor family, which we term IL-22R, functions as a second component together with CRF2-4 to enable *IL*-*22* signaling. *IL*-*22* does not bind the IL-10R. Cell lines were identified that respond to *IL*-*22* by activation of STATs 1, 3, and 5, but were unresponsive to IL-10. In contrast to IL-10, *IL*-*22* does not inhibit the production of proinflammatory cytokines by monocytes in response to LPS nor does it impact IL-10 function on monocytes, but it...

Chemical Name: CRF2-4 cytokine receptor; DNA-Binding Proteins; Interleukins; Ligands; Lipopolysaccharides; Receptors, Cytokine; Receptors, Interleukin; Tumor Necrosis Factor; interleukin-10 receptor; *interleukin*-*22*; *interleukin*-*22* receptor; Interleuki n-10

2/3,AB,KWIC/9 (Item 9 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09496691 95273341 PMID: 7753792

Mammary tumor suppression by transforming growth factor beta 1 transgene expression.

Pierce DF; Gorska AE; Chytil A; Meise KS; Page DL; Coffey RJ; Moses HL Department of Cell Biology, Vanderbilt University School of Medicine, Nashville, TN 37232-2175, USA.

Proceedings of the National Academy of Sciences of the United States of America (UNITED STATES) May 9 1995, 92 (10) p4254-8, ISSN 0027-8424 Journal Code: PV3

Contract/Grant No.: CA 42572, CA, NCI; CA 46413, CA, NCI; CA 48799, CA, NCI

Languages: ENGLISH

Document type: Journal Article

Record type: Completed

In cell culture, type alpha transforming growth factor (TGF-alpha) stimulates epithelial cell growth, whereas TGF-beta 1 overrides this stimulatory effect and is growth inhibitory. Transgenic mice that overexpress TGF-alpha under control of the mouse mammary tumor virus (MMTV) promoter/enhancer exhibit mammary ductal hyperplasia and stochastic development of mammary carcinomas, a process that

can be accelerated by administration of the chemical carcinogen 7,12-dimethylbenz[a]anthracene. MMTV-TGF-beta 1 transgenic mice display mammary ductal hypoplasia and do not develop mammary tumors. We report that in crossbreeding experiments involving the production of mice carrying both the MMTV-TGF-beta 1 and MMTV-TGF-alpha transgenes, there is marked suppression of mammary tumor formation and that MMTV-TGF-beta 1 transgenic mice are resistant to 7,12-dimethylbenz[a]anthracene-induced mammary tumor formation. These data demonstrate that overexpression of TGF-beta 1 in vivo can markedly suppress mammary tumor development.

Chemical Name: *Transforming* *Growth* *Factor* *beta*; *9*,10-Dimethyl-1,2-benzanthracene; Globins

2/3,AB,KWIC/10 (Item 10 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09266801 97207132 PMID: 9054625
The angiogenic switch in hamster buccal pouch keratinocytes is dependent on TGFbeta-1 and is unaffected by ras activation.
Lingen MW; DiPietro LA; Solt DB; Bouck NP; Polverini PJ
Northwestern University Medical School, Department of Pathology, Chicago, IL 60611, USA.
Carcinogenesis (ENGLAND) Feb 1997, 18 (2) p329-38, ISSN 0143-3334 Journal Code: C9T
Contract/Grant No.: K15 DE00313, DE, NIDCR; RO1 CA 52750, CA, NCI; RO1 CA 64239, CA, NCI; +
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
This study was undertaken to investigate the mechanisms by which Syrian hamster buccal pouch keratinocytes treated in vivo with 7,12-dimethylbenz[a]anthracene (DMBA), switch from an angio-inhibitory to an angiogenic phenotype. Cells were cultured from pouches at various times after exposure to carcinogen and their angiogenic activity assessed. The angio-inhibitory activity present in conditioned media from normal cells was lost as early as 3 weeks after carcinogen treatment, resulting in weak expression of angiogenic activity. By 5 weeks, cells had become strongly angiogenic due to the secretion of high levels of TGFbeta-1, a potent angiogenic factor. Because the switch to high levels of secreted TGFbeta-1 occurred at the same time as the activation of the H-ras oncogene, non-angiogenic cell lines lacking an activated H-ras oncogene were stably transfected with mutant H-ras and their transformed and angiogenic phenotypes were evaluated. Although ras transfection drove two of the three cultured cell lines to anchorage independence and modestly increased their ability to clone in low serum, it had no effect on the angiogenic phenotype or on the level of secreted active TGFbeta-1. These results demonstrate that the angiogenic phenotype in the hamster buccal pouch model of oral carcinogenesis develops in a step-wise fashion with an early decrease in the production of an inhibitor of angiogenesis and a subsequent marked increase in the secretion of the inducer TGFbeta-1. Although the activation of the H-ras oncogene contributed to anchorage independence, it did not affect the expression of the angiogenic phenotype in this model system.
Chemical Name: Carcinogens; *Transforming* *Growth* *Factor* *beta*; *9*,10-Dimethyl-1,2-benzanthracene

2/3,AB,KWIC/11 (Item 11 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09232601 97014240 PMID: 9157328
Identification of functional receptors for ciliary neurotrophic factor on chick ciliary ganglion neurons.
Koshlukova S; Finn TP; Nishi R; Halvorsen SW
Department of Biochemical Pharmacology, State University of New York at Buffalo, Buffalo, NY 14260, USA.
Neuroscience (UNITED STATES) Jun 1996, 72 (3) p821-32, ISSN 0306-4522 Journal Code: NZR
Contract/Grant No.: NS-25767, NS, NINDS; NS-30232, NS, NINDS Languages: ENGLISH
Document type: Journal Article
Record type: Completed
Ciliary neurotrophic factor and an avian homolog, growth promoting activity, are members of the cytokine/neurokine family of trophic factors and have been proposed to function as survival and developmental factors for ciliary ganglion neurons in vivo. Here we identify for the first time functional receptors for ciliary neurotrophic factor and growth promoting activity on cultured ciliary ganglion neurons. [(125)I]Rat ciliary neurotrophic factor binding studies indicate that rat ciliary neurotrophic factor and growth promoting activity bind to these receptors with a single affinity, while human ciliary neurotrophic factor recognizes both a high- and low-affinity site. Comparison of the relative potency of human ciliary neurotrophic factor and avian growth promoting activity in biological assays indicates that growth promoting activity is three to five times more active in promoting survival and in regulating acetylcholine receptors. The binding of ciliary neurotrophic factor is specific, sensitive to phosphatidylinositol-specific phospholipase C and partially inhibited by leukemia inhibitory factor, but not inhibited by other members of the human neurokine family, including interleukin-6, *interleukin*-*22* and oncostatin M. Cross-linking of [(125)I]rat ciliary neurotrophic factor to ciliary neurons results in the

specific labeling of three proteins with estimated molecular masses of 153,000, 81,000 and 72,000. Only the 81,000 molecular weight component is released from the cells after treatment with phosphatidylinositol-specific phospholipase C, suggesting a membrane attachment via a glycosylphosphatidylinositol linkage. Stimulation with ciliary neurotrophic factor or growth promoting activity, but not by other neurokines, results in the rapid tyrosine phosphorylation of a 90,000 molecular weight protein that is inhibited by pretreatment with phosphatidylinositol-specific phospholipase C. In conclusion, we report here the pharmacological and functional properties of ciliary neurotrophic factor receptors on embryonic ciliary ganglion neurons. These results provide the means for elaborating the molecular mechanisms of ciliary neurotrophic factor action and understanding its physiological role in a defined neuronal population.

... phosphatidylinositol-specific phospholipase C and partially inhibited by leukemia inhibitory factor, but not inhibited by other members of the human neurokine family, including interleukin-6, *interleukin*-*22* and oncostatin M. Cross-linking of [(125)I]rat ciliary neurotrophic factor to ciliary neurons results in the specific labeling of three proteins with estimated...

2/3,AB,KWIC/12 (Item 12 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08273659 95042313 PMID: 7954410
Lack of transforming growth factor-beta 1 expression in benign skin tumors of p53null mice is prognostic for a high risk of malignant conversion.
Cui W; Kemp CJ; Duffie E; Balmain A; Akhurst RJ
Department of Medical Genetics, University of Glasgow, Duncan Guthrie Institute, Yorkhill, United Kingdom.
Cancer research (UNITED STATES) Nov 15 1994, 54 (22) p5831-6, ISSN 0008-5472 Journal Code: CNF
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
Expression of transforming growth factor beta 1 (TGF beta 1) protein was examined in chemically induced benign skin tumors with genetically defined empirical risks for malignant conversion. Benign tumors induced in mice which have both alleles of the p53 gene deleted have a malignant conversion frequency of approximately 50%, whereas similar tumors induced in wildtype and heterozygous p53 mice have conversion probabilities of 3 and 8%, respectively (Kemp et al., Cell, 74: 813-822, 1993). The TGF beta 1 antibody, anti-CC (1-30-1), was shown to stain either the proliferative keratinocyte compartment of the tumor or the tumor stroma, whereas another TGF beta 1 antibody, anti-LC (1-30-1), stained highly differentiated granular cells of the tumors. A strong correlation was found between staining of the proliferative keratinocyte compartment of tumors with the anti-CC (1-30-1) antibody and tumor genotype. Only 18% (6 of 32) of homozygous p53 null tumors showed any basal keratinocyte staining with this antibody, whereas over 80% (32 of 38) of heterozygous and wild-type tumors showed positive staining. Additionally, in most tumors examined, the spatial distribution of staining for the proliferating cell nuclear antigen appeared to be mutually exclusive with that of TGF beta 1 on adjacent serial sections. This suggests that, in these cases, tumor keratinocytes are sensitive to negative growth regulation by TGF beta. TGF beta 1 protein staining in benign tumors is thus prognostic for a low probability of malignant conversion, and its expression may be mechanistically involved in limiting malignant conversion since, at the benign tumor stage examined, keratinocytes are still sensitive to growth inhibition by TGF beta 1.
Chemical Name: Proliferating Cell Nuclear Antigen; *Transforming* *Growth* *Factor* *beta*; *9*,10-Dimethyl-1,2-benzanthracen e

2/3,AB,KWIC/13 (Item 13 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08193214 94303148 PMID: 8030200
Detection of the readthrough protein of barley yellow dwarf virus. Cheng SL; Domier LL; D'Arcy CJ
Department of Plant Pathology, University of Illinois at Urbana-Champaign 61801.
Virology (UNITED STATES) Aug 1 1994, 202 (2) p1003-6, ISSN 0042-6822 Journal Code: XEA
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
The single open reading frame (ORF) 5 encoding the 50-kDa protein of barley yellow dwarf virus PAV-IL (BYDV-PAV-IL) was expressed in bacteria, purified, and used as an immunogen/antigen to produce/screen antibodies specific to the 50-kDa protein. Two monoclonal antibodies (MAb PAV-*IL* -*22* kDa and MAb PAV-IL-50 kDa) raised against BYDV-PAV-IL could specifically detect the presence of the 72-kDa readthrough protein in extracts from the BYDV-infected leaf tissue. The results suggest that ORF 5 (50-kDa protein) is translated by readthrough of ORF 3 (22-kDa coat protein) to produce the 72-kDa protein. The readthrough protein is thought to be a structural protein on the external surface of BYDV. ... expressed in bacteria, purified, and used as an immunogen/antigen to produce/screen antibodies specific to the 50-kDa

protein. Two monoclonal antibodies (MAb PAV-*IL*-*22* kDa and MAb PAV-IL-50 kDa) raised against BYDV-PAV-IL could specifically detect the presence of the 72-kDa readthrough protein in extracts...

2/3,AB,KWIC/14 (Item 14 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08109548 94128214 PMID: 8297479
Transforming growth factor-beta 1 expression in Syrian hamster cheek pouch carcinogenesis.
Zenklusen JC; Stockman SL; Fischer SM; Conti CJ; Gimenez-Conti IB University of Texas M. D. Anderson Cancer Center, Science Park-Research Division, Smithville 78957.
Molecular carcinogenesis (UNITED STATES) Jan 1994, 9 (1) p10-6, ISSN 0899-1987 Journal Code: AEQ
Contract/Grant No.: RR5511, RR, NCRR
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
The expression pattern of transforming growth factor-beta 1 (TGF-beta 1) during the stages of complete carcinogenesis in the hamster cheek pouch model was studied. The right cheek pouches of 18 male hamsters were treated with 0.5%, 7,12-dimethylbenz[a]anthracene (DMBA) for 16 wk. TGF-beta 1 was detected immunohistochemically in the resulting samples with two different polyclonal monospecific antibodies that recognize intracellular and extracellular forms of TGF-beta 1. In the normal cheek pouch, extracellular protein stained the corium strongly, but the reaction was not evenly distributed. As treatment progressed, the reaction increased in both area and intensity; the peak was reached at 8 wk. Intracellular TGF-beta 1 expression followed a similar pattern, with a peak at 4 wk of treatment. The results of northern blot analysis were concordant with the immunohistochemical results. Overexpression of TGF-beta 1 was also observed in the malignant tumors, but only the extracellular form of the protein was present; intracellular TGF-beta 1 was not detected in these tumors. The expression of TGF-beta 1 in this carcinogenesis model seems to have two formal stages, the first being an overexpression step as a reaction to the uncontrolled growth and the second being one in which tumors have no internal expression of TGF-beta 1 but in which external protein accumulates in the surrounding stroma. A possible explanation of this paradox may be that TGF-beta 1 has functions other than its growth-repressing activity.
Chemical Name: RNA, Messenger; *Transforming* *Growth* *Factor* *beta*; *9*,10-Dimethyl-1,2-benzanthracene

2/3,AB,KWIC/15 (Item 15 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07637745 92369012 PMID: 1504019
Discordant transforming growth factor beta 1 RNA and protein localization during chemical carcinogenesis of the skin.
Fowlis DJ; Flanders KC; Duffie E; Balmain A; Akhurst RJ
Duncan Guthrie Institute of Medical Genetics, University of Glasgow, Yorkhill Hospitals, United Kingdom.
Cell growth & differentiation (UNITED STATES) Feb 1992, 3 (2) p81-91 , ISSN 1044-9523 Journal Code: AYH
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
Transforming growth factor beta (TGF-beta) inhibits proliferation of normal keratinocytes, and this response is retained, to variable extents, in benign tumors of the skin (S. Haddow, D. J. Fowlis, K. Parkinson, R. J. Akhurst, and A. Balmain, Oncogene, 6: 1465-1470, 1991). To investigate the profile of TGF-beta biosynthesis during various stages of chemical carcinogenesis of the skin, we used a combination of ribonuclease protection assay, in situ hybridization with gene-specific probes for TGF-beta 1, -beta 2, and -beta 3, and immunohistochemistry with isoform-specific antibodies against TGF-beta 1. Following 12-O-tetradecanoylphorbol-13-acetate treatment of adult mouse skin, there was a rapid induction of TGF-beta 1 protein. Intracellular TGF-beta 1 protein was localized to suprabasal keratinocytes, and the extracellular form was localized predominantly to the dermis. Despite ubiquitous induction of TGF-beta 1 protein by 12-O-tetradecanoylphorbol-13-acetate in various mouse strains, we noted strain-specific differences in the quantitative induction of TGF-beta 1 RNA. Papillomas and carcinomas induced in vivo had elevated levels of TGF-beta 1 RNA within the basal keratinocyte compartment but did not contain significant levels of TGF-beta 1 protein within the tumor. We postulate that the tumor evades TGF-beta 1-controlled negative growth regulation by altered translational and/or posttranslational processing mechanisms of this growth factor. Levels of TGF-beta 2 and -beta 3 RNA were not elevated at any stage of chemical carcinogenesis of the skin.

Chemical Name: Phorbol Esters; RNA, Messenger; *Transforming* *Growth* *Factor* *beta*; *9*,10-Dimethyl-1,2-benzanthracen e

2/3,AB,KWIC/16 (Item 16 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07559774 92097106 PMID: 1728410
Eosinophils, tissue eosinophilia, and eosinophil-derived transforming growth factor alpha in hamster oral carcinogenesis.
Ghiabi M; Gallagher GT; Wong DT
Department of Oral Medicine and Oral Pathology, Harvard School of Dental Medicine, Boston, Massachusetts 02115.
Cancer research (UNITED STATES) Jan 15 1992, 52 (2) p389-93, ISSN 0008-5472 Journal Code: CNF
Contract/Grant No.: DE00318, DE, NIDCR; DE08680, DE, NIDCR Languages: ENGLISH
Document type: Journal Article
Record type: Completed
Eosinophilia in tissues and/or circulating blood is known to be associated with a wide variety of malignancies but the role of the eosinophil in neoplastic conditions is not known. Using the cheek pouch of the Syrian hamster as an experimental model for oral carcinogenesis, it has recently been shown that eosinophils at sites of developing oral cancer express the multifunctional cytokine, transforming growth factor alpha (TGF-alpha). This study investigated the time course of eosinophil infiltration, tissue eosinophilia associated with malignant epithelium, and eosinophil-derived TGF-alpha mRNA during the 16-week 7,12-dimethylbenz[a]anthracene (DMBA)-induced oral cancer development process. The results reveal that the occasional eosinophil is normally present in the lamina propria of hamster oral mucosa. With progressive DMBA treatments, there is an increase of eosinophils infiltrating into the lamina propria. By weeks 12-16, the number of eosinophils is significantly higher in DMBA-treated pouches than in control pouches treated with the vehicle mineral oil alone. Analysis of the infiltrating eosinophils into fully developed hamster oral carcinomas reveals that tissue eosinophilia is associated with 78% of the stromal areas associated with malignant epithelium, while only 7% of sites associated with non-tumor oral epithelium (normal, hyperplastic-dysplastic) exhibited eosinophilia. Furthermore, the majority of the eosinophils associated with malignant epithelium were found to contain TGF-alpha mRNA. The number of TGF-alpha mRNAs containing eosinophils associated with malignant oral epithelium is significantly higher than that associated with nonmalignant oral epithelium. Together, these results suggest that eosinophils are recruited to tumor-developing sites, that they predominantly associate with malignant epithelium, and that most tumor-associated eosinophils express the cytokine TGF-alpha.

Chemical Name: RNA, Messenger; *Transforming* *Growth* *Factor* *beta*; *9*,10-Dimethyl-1,2-benzanthracene

2/3,AB,KWIC/17 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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10109525 Genuine Article#: 480UY Number of References: 0 Title: Identification, cloning and characterization of a novel soluble receptor which binds *IL*-*22* and neutralizes its activity Author(s): Kotenko SV; Izotova LS; Mirochnitchenko OV; Dickensheets H; Donnelly RP; Pestka S Corporate Source: Univ Med & Dent New Jersey,New Jersey Med Sch, Dept Biochem & Mol Biol,Newark//NJ/07103; Univ Med & Dent New Jersey,Robert Wood Johnson Med Sch, Dept Mol Genet & Microbiol,Newark//NJ/07103; US FDA,Div Therapeut Proteins,Rockville//MD/20857
Journal: JOURNAL OF LEUKOCYTE BIOLOGY, 2001, S, P26-26
ISSN: 0741-5400 Publication date: 20010000
Publisher: FEDERATION AMER SOC EXP BIOL, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814-3998 USA
Language: English Document Type: MEETING ABSTRACT

Title: Identification, cloning and characterization of a novel soluble receptor which binds *IL*-*22* and neutralizes its activity

2/3,AB,KWIC/18 (Item 2 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2001 Inst for Sci Info. All rts. reserv.

10109524 Genuine Article#: 480UY Number of References: 0 Title: *IL*-*22* is a tightly regulated IL-10-like molecule that induces an acute-phase response and renal tubular basophilia. Author(s): Fouser LA; Lambert AJ; Clark E; Deng BJ; Tan XY; Spaulding V; Wang IM; Kobayashi M; Whitters M; Thibodeaux D; Leonard J; Ling V; Wu P ; Annis B; Lu ZJ; Zollner R; Jacobs K; Goad B; Pittman D Corporate Source: Genet Inst Inc,Cambridge//MA/02140
Journal: JOURNAL OF LEUKOCYTE BIOLOGY, 2001, S, P26-26
ISSN: 0741-5400 Publication date: 20010000
Publisher: FEDERATION AMER SOC EXP BIOL, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814-3998 USA
Language: English Document Type: MEETING ABSTRACT

Title: *IL*-*22* is a tightly regulated IL-10-like molecule that induces an acute-phase response and renal tubular basophilia.

2/3,AB,KWIC/19 (Item 3 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2001 Inst for Sci Info. All rts. reserv.

10045395 Genuine Article#: 479FW Number of References: 34 Title: A novel, soluble homologue of the human IL-10 receptor with preferential expression in placenta (ABSTRACT AVAILABLE) Author(s): Gruenberg BH; Schoenemeyer A; Weiss B; Toschi L; Kunz S; Wolk K; Asadullah K; Sabat R (REPRINT)
Corporate Source: Schering AG,Dept Expt Dermatol,Muellerstr 178/D-13342 Berlin//Germany/ (REPRINT); Schering AG,Dept Expt Dermatol,D-13342 Berlin//Germany/; Schering AG,Enabeling Technol Genom & Bioinformat,D-13342 Berlin//Germany/; Humboldt Univ,Med Sch Charite, Inst Med Immunol,D-10098 Berlin//Germany/
Journal: GENES AND IMMUNITY, 2001, V2, N6 (OCT), P329-334
ISSN: 1466-4879 Publication date: 20011000
Publisher: NATURE PUBLISHING GROUP, HOUNDMILLS, BASINGSTOKE RG21 6XS, HAMPSHIRE, ENGLAND
Language: English Document Type: ARTICLE
Abstract: The cytokine receptor family type 2 (CRF2) comprises receptors for important immunomediators like interferons and interleukin-10 (IL-10). We identified a novel member of this family which represents the first exclusively soluble receptor in this group and was therefore designated as CRF2-soluble 1 (CRF2-s1). The CRF2-s1 gene covers about 28 kb and is located on chromosome 6 in close proximity to the CRF2 members interferon (IFN)-gamma receptor 1 and IL-20 receptor 1. It comprises seven exons and generates two different mRNA splice variants, CRF2-s1-long and CRF2-s1-short. CRF2-s1-long and CRF2-s1-short encode proteins of 263 and 231 amino acids, respectively. A comparison of predicted protein structures led to the postulation that each receptor variants binds a different ligand. Quantitative analysis of human mRNA expression revealed a very restricted pattern for both splice forms. CRF2-s1 turned out to be the first member of this receptor family which was expressed neither in resting nor in stimulated leucocyte populations. CRF2-s1-long was only expressed in placenta, whereas CRF2-s1-short was additionally expressed in human mammary gland and, at a lower level, in skin, spleen, thymus and stomach. The preferential expression of CRF2-s1 in placenta suggests a role for this receptor in establishing and maintaining successful pregnancy.

2/3,AB,KWIC/20 (Item 4 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2001 Inst for Sci Info. All rts. reserv.

09596468 Genuine Article#: 410TA Number of References: 0 Title: Human *IL*-*22* (IL-TIF) is a novel homolog of IL-10 that phosphorylates STAT 3 in Colon carcinoma cells expressing the IL-22R1 chain
Author(s): Nagalakshmi ML; Parham C; Rascle A; Menon S; Moore K; Malefyt RD Corporate Source: DNAX Res Inst Mol & Cellular Biol Inc,Palo Alto//CA/94304 Journal: FASEB JOURNAL, 2001, V15, N5,2 (MAR 8), PA1052-A1052 ISSN: 0892-6638 Publication date: 20010308
Publisher: FEDERATION AMER SOC EXP BIOL, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814-3998 USA
Language: English Document Type: MEETING ABSTRACT

Title: Human *IL*-*22* (IL-TIF) is a novel homolog of IL-10 that phosphorylates STAT 3 in Colon carcinoma cells expressing the IL-22R1 chain

2/3,AB,KWIC/21 (Item 5 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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08756154 Genuine Article#: 326BW Number of References: 24 Title: Deletion of the Alu-VpA/MycL1 (1p34.3) locus is a negative prognostic sign in human colorectal cancer (ABSTRACT AVAILABLE) Author(s): Kashkin KN (REPRINT) ; Perevoschikov AG; Nikolaev AV; Turbin DA; Fleischman EW
Corporate Source: RUSSIAN ACAD MED SCI,BLOKHIN CANC RES CTR/MOSCOW 115478//RUSSIA/ (REPRINT)
Journal: MOLECULAR BIOLOGY, 2000, V34, N3 (MAY-JUN), P337-344 ISSN: 0026-8933 Publication date: 20000500
Publisher: CONSULTANTS BUREAU, 233 SPRING ST, NEW YORK, NY 10013 Language: English Document Type: ARTICLE
Abstract: We examined deletions of the short arm of chromosome 1 and aberrations of the microsatellite locus Alu-VpA/MycL1 (1p34.3) in human primary colorectal adenocarcinomas. Cytogenetically discernible deletions in Ip were found in 45% (14/31) of informative tumors. The 1p- tumors commonly exhibited a polyploid karyotype (Fisher P-1 = 0.023) and a larger number of rearranged chromosomes (P-2 = 0.045) versus those without Ip deletions. The Ip deletions often combined with chromosome 5 monosomy (chi(2) = 6.24; p = 0.013), chromosome 15 monosomy (chi(2) = 4.20; p = 0.040), and 11q deletions (P-2 = 0.035). Among the 50 carcinomas, *Il* (*22*%) showed Alu VpA/MycL1 instability, and 14% (6/43 informative) had lost the Alu-VpA/MycL1 allele. The genetic alterations thus revealed were collated with the clinical and morphological features of the tumors. The loss of the Ip material was shown to be correlated with marked karyotype aberrations in colorectal rumors, and Alu-VpA/MycL1 allele deletions were tightly associated with relapses or metastasis within 30 months after surgery.
...Abstract: 24; p = 0.013), chromosome 15 monosomy

(chi(2) = 4.20; p = 0.040), and 11q deletions (P-2 = 0.035). Among the 50 carcinomas, *Il* (*22*%) showed Alu VpA/MycL1 instability, and 14% (6/43 informative) had lost the Alu-VpA/MycL1 allele. The genetic alterations thus revealed were collated with...

2/3,AB,KWIC/22 (Item 6 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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08290062 Genuine Article#: 266VV Number of References: 63 Title: Effects of dietary omega-3 and omega-6 lipids and vitamin E on serum cytokines, lipid mediators and anti-DNA antibodies in a mouse model for rheumatoid arthritis (ABSTRACT AVAILABLE)
Author(s): Venkatraman JT (REPRINT) ; Chu WC
Corporate Source: SUNY BUFFALO,DEPT PHYS THERAPY EXERCISE & NUTR SCI, NUTR PROGRAM, 15 FARBER HALL/BUFFALO//NY/14214 (REPRINT)
Journal: JOURNAL OF THE AMERICAN COLLEGE OF NUTRITION, 1999, V18, N6 (DEC) , P602-613
ISSN: 0731-5724 Publication date: 19991200
Publisher: AMER COLL NUTRITION, C/O HOSP. JOINT DIS. 301 E. 17TH ST., NEW YORK, NY 10003
Language: English Document Type: ARTICLE
Abstract: Objective: Omega-3 (omega-3) fatty acid rich-fish oil (FO) and vitamin E (vit-E) may delay the progress of certain autoimmune diseases. The present study examined the mechanism of action of omega-3 and omega-6 lipids and vit-E on the serum cytokines and lipid mediators in autoimmune-prone MRL/1pr mice (a model for rheumatoid arthritis, RA). The 1pr (lymphoproliferative) gene is overexpressed in these mice causing extensive lymphoproliferation, lupus-like symptoms and accelerated aging.

Methods: Weanling female MRL/lpr and congenic control MRL/+ + mice were fed 10% corn oil (CO, omega 6) or FO-based semipurified diets containing two levels of vitamin E (vit-E-75, I.U. and vit-E-500 I.U/Kg diet) for four months. At the end of the experiment, serum anti-DNA antibodies, cytokines and lipid mediators levels were determined.

Results: The appearance of enlarged lymph nodes was delayed in the mice fed FO, and the FO-500 IU vit-E diet offered further protection against enlargement of lymph nodes. The MRL/lpr mice exhibited significantly higher levels of serum anti-dsDNA antibodies. The FO-fed mice had significantly lower serum IL-6, IL-IO, IL-12, TNF-alpha PGE(2), TXB2 and LTB4 levels compared with GO-fed mice. In mice fed 500 TU vit-E diets, the serum IL-6, IL-10, IL-12 and TNF-alpha levels were significantly lower and serum IL-1 beta was significantly higher compared to 75 IU-vit-E-fed mice in CO/FO or both. The levels of anti-DNA antibodies, IL-4, IL-6, TNF-alpha, IL-10 and IL-12 were higher in the sera of MRL/lpr mice. The FO diet lowered the levels of these cytokines (except IL-4) and lipid mediators. Adding 500 IU of vit-E to the FO diet further lowered the levels of IL-6, IL-10, *IL*- *22*, and TNF-alpha.

Conclusion: ft is clear from our observations that the beneficial effects of FO can be enhanced by the addition of 500 IU of vit-E in the diet The FO diet containing 500 IU of vit-E may specifically modulate the levels of IL-6, IL-10, IL-12 and TNF-alpha and thereby may delay the onset of autoimmunity in the MRL/lpr mouse model. The observations from this study may form a basis for selective nutrition intervention based on specific fatty acids and antioxidants in delaying the progress of RA.

...Abstract: except IL-4) and lipid mediators. Adding 500 IU of vit-E to the FO diet further lowered the levels of IL-6, IL-10, *IL*- *22*, and TNF-alpha.

Conclusion: ft is clear from our observations that the beneficial effects of FO can be enhanced by the addition of 500 IU...

2/3,AB,KWIC/23 (Item 7 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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07757480 Genuine Article#: 205BT Number of References: 76 Title: p38 alpha mitogen-activated protein kinase is activated by CD28-mediated signaling and is required for IL-4 production by human CD4(+)CD45RO(+) T cells and Th2 effector cells (ABSTRACT AVAILABLE) Author(s): Schafer PH; Wadsworth SA; Wang LW; Siekierka JJ (REPRINT)
Corporate Source: RW JOHNSON PHARMACEUT RES INST,DRUGS DISCOVERY RES, ROUTE 202, POB 300/RARITAN//NJ/08869 (REPRINT); RW JOHNSON PHARMACEUT RES INST,DRUGS DISCOVERY RES/RARITAN//NJ/08869
Journal: JOURNAL OF IMMUNOLOGY, 1999, V162, N12 (JUN 15), P7110-7119 ISSN: 0022-1767 Publication date: 19990615
Publisher: AMER ASSOC IMMUNOLOGISTS, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814
Language: English Document Type: ARTICLE
Abstract: T cell proliferation and cytokine production usually require stimulation via both the TCR/CD3 complex and the CD28 costimulatory receptor, Using purified human CD4(+) peripheral blood T cells, we show that CD28 stimulation alone activates p38 alpha mitogen;activated protein kinase (p38 alpha), Cell proliferation induced by CD28 stimulation alone, a response attributed to CD4(+)CD45RO(+) memory T cells, was blocked by the highly specific. p38 inhibitors

SE 203580 (IC50 = 10-80 nM) and RWJ 67657 (IC50 = 0.5-4 nM). In contrast, proliferation induced by anti-CD3 plus anti-CD28 mAbs was not blocked, Inhibitors of p38 also blocked CD4(+) T cell production of IL-4 (SB 203580 IC50 = 24-100 nM), but not *IL*-*22* in response to CD3 and CD28 stimulation, IL-5, TNF-alpha, and IFN-gamma production were also inhibited, but to a lesser degree than IL-4, IL-4 production was attributed to CD4(+)CD45RO(+) T cells, and its induction was suppressed by p38 inhibitors at the mRNA level. In polarized Th1 and Th2 cell lines, SE 203580 strongly inhibited IL-4 production by Th2 cells (IC50 = 10-80 nM),but only partially inhibited IFN-gamma and IL-2 production-by Th1 cells (<50% inhibition at 1 mu M). In both Th1 and Th2 cells, CD28 signaling activated p38 alpha and was required for cytokine production. These results show that p38 alpha plays an important role in some, but not all, CD28-dependent cellular responses. Its preferential involvement in IL-4 production by CD4(+)CD45RO(+) T cells and Th2 effector cells suggests that p38 alpha may be important in the generation of Th2-type responses in humans.

...Abstract: CD28 mAbs was not blocked, Inhibitors of p38 also blocked CD4(+) T cell production of IL-4 (SB 203580 IC50 = 24-100 nM), but not *IL*-*22* in response to CD3 and CD28 stimulation, IL-5, TNF-alpha, and IFN-gamma production were also inhibited, but to a lesser degree than IL...

2/3,AB,KWIC/24 (Item 8 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

07262879. Genuine Article#: 142VE Number of References: 24 Title: Induction of tyrosinase-reactive T cells by treatment with dacarbazine, cisplatin, interferon-alpha +/- interleukin-2 in patients with metastatic melanoma (ABSTRACT AVAILABLE)
Author(s): Schmittel A (REPRINT) ; Keilholz U; Max R; Thiel E; Scheibenbogen C
Corporate Source: FREE UNIV BERLIN,KLINIKUM BENJAMIN FRANKLIN, MED KLIN 3, HINDENBURGDAMM 30/D-12200 BERLIN//GERMANY/ (REPRINT); UNIV HEIDELBERG,MED KLIN & POLIKLIN HAMATOL ONKOL & RHEUMATOL 5/HEIDELBERG//GERMANY/
Journal: INTERNATIONAL JOURNAL OF CANCER, 1999, V80, N1 (JAN 5), P39-43 ISSN: 0020-7136 Publication date: 19990105
Publisher: WILEY-LISS, DIV JOHN WILEY & SONS INC, 605 THIRD AVE, NEW YORK, NY 10158-0012
Language: English Document Type: ARTICLE
Abstract: We have shown the presence of tyrosinase-reactive T cells in the peripheral blood of melanoma patients, who had been in remission after treatment with *IL*-*22*-containing regimens. In this consecutive study, we analyzed the T cell response to various peptides derived from tyrosinase in serial blood samples obtained from 7 stage-IV melanoma patients before, during and following treatment, All patients were treated within a randomized trial (EORTC 18951) with cisplatin (CDDP), dacarbazine (DTIC), interferon-alpha (IFN-alpha) +/- interleukin-2 (IL-2), Using an ELISPOT assay detecting peptide-specific IFN-gamma release, we measured the T-cell response to 4 different HLA class I-binding peptide epitopes derived from tyrosinase containing an HLA-A2.1-, HLA-A24- or HLA-B44-binding motif in peripheral-blood mononuclear cells (PBMC). In one patient, tyrosinase-reactive T cells were detected before therapy. In 4 out of 7 patients, tyrosinase-reactive T cells against both HLA-A2.1-binding peptides and the B44-binding peptide became detectable at frequencies of up to 30 in 5 x 10(5) lymphocytes following treatment, These patients received CDDP, DTIC and IFN-alpha, 2 of them without IL-2 and 2 with IL-2, resulting in one complete remission and 3 partial remissions. Two patients relapsed 8 and 9 months after treatment. At the time of relapse, no T cells reactive with tyrosinase were detectable. Our results show that high frequencies of tyrosinase-reactive T cells in the peripheral blood of melanoma patients can be induced by chemotherapy in combination with IFN-alpha, regardless of concomitant IL-2 administration. (C) 1999 Wiley-Liss, Inc.

Abstract: We have shown the presence of tyrosinase-reactive T cells in the peripheral blood of melanoma patients, who had been in remission after treatment with *IL*-*22*-containing regimens. In this consecutive study, we analyzed the T cell response to various peptides derived from tyrosinase in serial blood samples obtained from 7...

2/3,AB,KWIC/25 (Item 9 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

07123330 Genuine Article#: 125ZF Number of References: 20 Title: Effect of SiGe thickness on crystallisation and electrical properties of sputtered silicon film in Si/SiGe/insulator structure (ABSTRACT AVAILABLE)
Author(s): Jelenkovic EV (REPRINT) ; Tong KY
Corporate Source: HONG KONG POLYTECH UNIV,DEPT ELECT ENGN/HONG KONG//PEOPLES R CHINA/ (REPRINT)
Journal: APPLIED SURFACE SCIENCE, 1998, V135, N1-4 (SEP), P143-149 ISSN: 0169-4332 Publication date: 19980900

Publisher: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE AMSTERDAM, NETHERLANDS Language: English Document Type: ARTICLE
Abstract: Layered structures Si/SiGe were deposited on silicon oxide by RF sputtering system and were furnace crystallised at a temperature of 550 degrees C. The effect of SiGe seeding layer thickness on crystallisation and electrical properties of the top silicon film was studied for SiGe films with thicknesses of *Il*, *22* and 45 nm. Crystallisation process was characterised by scanning electron microscopy (SEM) and X-ray diffraction (XRD). Doping of stacked structures by phosphorous and boron was investigated through measurement of sheet resistance and Hall mobility. In the scope of investigated thickness ranges, Ii nm thick seeding layer showed the best performance. It is effective in reducing the crystallisation time of the top silicon film, while providing improved morphological and electrical properties of the stacked structure. (C) 1998 Elsevier Science B.V. All rights reserved.

...Abstract: The effect of SiGe seeding layer thickness on crystallisation and electrical properties of the top silicon film was studied for SiGe films with thicknesses of *Il*, *22* and 45 nm. Crystallisation process was characterised by scanning electron microscopy (SEM) and X-ray diffraction (XRD). Doping of stacked structures by phosphorous and boron...

2/3,AB,KWIC/26 (Item 10 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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06471980 Genuine Article#: YV763 Number of References: 28 Title: The peritoneal fluid levels of interleukin-12 in women with endometriosis (ABSTRACT AVAILABLE)
Author(s): Zeyneloglu HB; Senturk LM; Seli E; Bahtiyar OM; Olive DL; Arici A (REPRINT)
Corporate Source: YALE UNIV,SCH MED, DEPT GYNECOL & OBSTET, DIV REPROD ENDOCRINOL, 333 CEDAR ST/NEW HAVEN//CT/06520 (REPRINT); YALE UNIV,SCH MED, DEPT GYNECOL & OBSTET, DIV REPROD ENDOCRINOL/NEW HAVEN//CT/06520
Journal: AMERICAN JOURNAL OF REPRODUCTIVE IMMUNOLOGY, 1998, V39, N2 (FEB) , P152-156
ISSN: 8755-8920 Publication date: 19980200
Publisher: MUNKSGAARD INT PUBL LTD, 35 NORRE SOGADE, PO BOX 2148, DK-1016 COPENHAGEN, DENMARK
Language: English Document Type: ARTICLE
Abstract: PROBLEM: Interleukin-12 (*IL*-*22*) is produced mainly by monocytes/macrophages, and it induces proliferation and cytotoxicity of T-cells and natural killer cells. In women with endometriosis, natural killer cell activity in the peritoneal fluid is significantly decreased. We aimed to measure the peritoneal fluid level of IL-12 in endometriosis.

METHOD OF STUDY:We measured 1L-12 levels in peritoneal fluid samples from women with or without endometriosis and in supernatants from endometrial stromal, ovarian stromal, and mesothelial cell cultures, using a high-sensitivity enzyme-linked immunosorbent assay.

RESULTS: The median concentration of IL-12 in the peritoneal fluid of women with endometriosis was 1.1 pg/ml (range, 0.2-5.5) and was 1.6 pg/ml (range, 0.4-2.8) in women without endometriosis, not a statistically significant difference. IL-12 was not detected in the supernatants of endometrial stromal, ovarian stromal, and mesothelial cell cultures.

CONCLUSION: Concentrations of IL-12 in the peritoneal fluid of women with or without endometriosis are low, but they are detectable and are not affected significantly by the presence of endometriosis.
Abstract: PROBLEM: Interleukin-12 (*IL*-*22*) is produced mainly by monocytes/macrophages, and it induces proliferation and cytotoxicity of T-cells and natural killer cells. In women with endometriosis, natural killer...

2/3,AB,KWIC/27 (Item 1 from file: 94)
DIALOG(R)File 94:JICST-EPlus
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00976698 JICST ACCESSION NUMBER: 90A0201872 FILE SEGMENT: JICST-E Effects of electrodeposition factors on the limiting current density of aluminum electroplating.
KATO YOSHIO (1); TATANO MASAYOSHI (1); TAKAHASHI SETSUKO (2) (1) NISSHINSEIKO TEKKOKEN; (2) Nisshin Steel Co., Ltd., New Materials Lab. Nisshin Seiko Giho(Nisshin Steel Technical Report), 1989, NO.61, PAGE.44-53 , FIG.16, TBL.2, REF.17
JOURNAL NUMBER: F0232AAC ISSN NO: 0387-2327
UNIVERSAL DECIMAL CLASSIFICATION: 621.793.3+621.357.7
LANGUAGE: Japanese COUNTRY OF PUBLICATION: Japan
DOCUMENT TYPE: Journal
ARTICLE TYPE: Original paper
MEDIA TYPE: Printed Publication
ABSTRACT: An electroplating process for aluminum using a butylpyridinium chloride AlCl3 molten salt bath has been developed. The effect of the bath temperature, flow rate, electrode length and line speed on the limiting current density were studied. The results obtained are summarized as follows: (1) The results

from the use of an electrolytic circuit apparatus show that the limiting current density is affected by the bath temperature, flow rate and electrode length. The following equations were obtained. iL=13.9u0.71l-0.63 (40.DEG.C.) *iL*= *22*.2u0.75l-0.67 (50.DEG.C.) iL=25.0u0.63l-0.55 (60.DEG.C.) Where iL: Limiting current density (A/dm2) u: Flow rate (m/sec) l: Electrode length (cm) (2) The results from the use of a continuous pilot plating line are summarized in the following equation. iL .VAR. u0.7l-0.6 (60.DEG.C.) This is in agreement with the equations obtained from the electrolytic circuit apparatus. (3) The morphology of the electroplated Al layer is also studied in this report. (auther abst.)
...ABSTRACT: is affected by the bath temperature, flow rate and electrode length. The following equations were obtained. iL=13.9u0.71l-0.63 (40.DEG.C.) *iL*=*22*.2u0.75l-0.67 (50.DEG.C.) iL=25.0u0.63l-0.55 (60.DEG.C.) Where iL: Limiting current density (A/dm2) u: Flow...
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